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ABSTRACT

Two nuclear accidents involving a plutonium sphere just subcritical in size occurred at the Los Alamos Laboratory, LA-1 in 1945 and LA-2 in 1946. Each of the so-called criticality experiments called for neutron reflecting material to be placed around the sphere. When enough of the neutrons which had leaked from the sphere were reflected back into the sphere to allow a self-sustaining chain of fission reactions, the assembly was said to be in the critical state. Because remote control devices were deemed unreliable at the time, the tamper material (tungsten carbide bricks in LA-1 and beryllium hemispheres in LA-2) was added by hand with the operator standing next to the assembly. In each case the critical size of the assembly was accidentally exceeded and the resultant exponentially increasing chain reaction emitted a burst of neutrons and gamma rays.

Ten persons were exposed to the radiation bursts which were largely composed of neutrons. The doses ranged from fatal in the case of the two operators, to small in the case of some survivors.

The two operators died within weeks as a result of acute radiation injury. Only six of the eight survivors were available for follow-up study ten or more years after the accident. Four of these six survivors are now dead, but the two living survivors are in excellent health with no clinical or laboratory evidence of late radiation injury. Two of the deceased died of acute myelogenous leukemia, another died at age 83 of refractory anemia, and the fourth of myocardial infarction. The heart attack could have been precipitated by the myxedema assumed to have been the result of the radiation exposure.

Within ten months of the end of World War II, two serious nuclear criticality accidents occurred at the Los Alamos Laboratory (Site Y) of the then Manhattan Project. Each accident resulted in the death of the operator and in the overexposure to ionizing radiations of eight other participants. Both accidents were the result of uncontrolled nuclear chain reactions in so-called critical assembly experiments using metallic plutonium. Because of the nature of the times at the end of World War II, the early published accounts gave little detailed information of the nature of the accidents or of how they occurred. In the present report, we

describe the now non-classified physical aspects of the accidents and bring up to date the medical histories of the survivors, last published in 1951, to show what has happened in the almost 35 years since the exposures occurred.

The criticality experiments were designed to provide information about the fission characteristics of metallic plutonium. The experiments were carried out with a sub-critical plutonium sphere exposed to neutrons from a small externally placed neutron source. Because of the size and shape of the sphere, so much neutron leakage occurred from the sphere that in the absence of neutron reflecting material, a fission chain reaction could not be sustained. By surrounding the sphere with the right amount of tamper (neutron reflecting) material, it was possible to reflect enough neutrons back into the sphere to achieve a delayed critical state. In this state, a constant rate of self-sustaining nuclear fission chain reactions could be achieved with or without extraneous neutrons. In the prompt critical state, the neutron reflection back into the sphere is great enough to sustain criticality even in the absence of delayed neutrons. When prompt criticality was exceeded, as i believed to have occurred in both accidents, fissioning of plutonium atoms increased exponentially with the release of a burst of neutrons and gamma rays.

Although the potential danger in this type of experimentation was fully appreciated, the physicists elected to carry out the procedure manually rather than remotely since a reliable remote control mechanism was not available at the time. The scientists in charge feared that failure of such a mechanism could lead to a minor but still serious nuclear explosion while manual operators, by meticulous attention to detail, could carry out the experiments with very little radiation exposure even while standing

next to the critical assembly. Hundreds of such experiments were carried out properly with negligible exposures of the operators.

In two experiments at Los Alamos after World War II, the critical state was accidentally exceeded. In these two accidents, ten experimenters were seriously irradiated; two of them died within weeks of their exposures. The fatal accidents and a brief description of how they occurred were published in a monograph in 1952.[1] That report focused primarily on the acute illness of the two fatally stricken operators and of seven of the eight survivors, all of whom received substantial doses of neutrons and gamma rays. The eighth survivor refused to consent to the follow-up study. Although all seven survivors have been followed periodically since 1950, their medical courses have not been published in the open literature. In the present report we bring up to date the case histories of seven of the eight survivors of the two Los Alamos accidents.

Description of the Accidents

In both accidents, the uncontrolled chain reactions occurred in a nickel-plated plutonium sphere.[2] The plutonium sphere in both accidents weighed 6.19 kilograms. In the first accident, designated LA-1, the tamper material used to surround the sphere was tungsten carbide (WC) bricks, while that in the second accident, designated LA-2, was beryllium in the form of hemispheres. On both occasions there was a prompt supercritical excursion believed to have been caused by the increased number of neutrons reflected back into the sphere by the tamper material. During the period of the exponentially increasing chain reaction, the assembly was engulfed by a clearly visible blue glow of intensity ionized air caused by electrons and soft x-rays. In the LA-1 accident, which occurred at night in a well illuminated laboratory, the flash lit up a newspaper being read by a

security guard 10-12 feet away with his back to the assembly (Case 2). In LA-2, in a sun-lit room, the glow was seen by five of the seven persons facing the assembly. Data from other published experiments on high intensity radiation sources suggest that a particle beam does not become visible until the radiation intensity is of the order of 6×10^7 R/second.[3]

In the LA-1 accident,[4] tungsten carbide (WC) bricks, each weighing 4.4 kilograms, were stacked around the carefully machined sphere. Two similar but slightly modified experiments with WC bricks had been successfully carried out the day of the accident. The fatal experiment scheduled for the next morning called for five bricks of WC on each side. Instead of waiting until the scheduled time, the physicist-operator (Case 1) began the experiment on the night of August 21, 1945 at 9:55 p.m. with only a military guard (Case 2) in the laboratory. While the guard was seated at a desk 10-12 feet away the operator added the WC bricks to the assembly. Four layers of bricks were in place and the final brick for the fifth layer was being carried to its place when the operator noted that the neutron flux was increasing rapidly. He attempted to withdraw the final brick (Figure 1) but it slipped out of his hand and fell onto the center of the assembly. The operator immediately pushed the final brick off the assembly with his right hand and dismantled the assembly to the point shown in Figure 2. The operator estimated that he remained in the vicinity of the reactor for at least ten minutes.[3] When seen at the hospital 30 minutes after the accident, he complained of numbness and tingling of his swollen hands.

In the LA-2 accident on May 21, 1946,[5,6] the physicist in charge (Case 3) who was about to leave for the Bikini test was indoctrinating his

security guard 10-12 feet away with his back to the assembly (Case 2). In LA-2, in a sun-lit room, the glow was seen by five of the seven persons facing the assembly. Data from other published experiments on high intensity radiation sources suggest that a particle beam does not become visible until the radiation intensity is of the order of 6×10^7 R/second.[3]

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In the LA-2 accident on May 21, 1946,[5,6] the physicist in charge (Case 3) who was about to leave for the Bikini test was indoctrinating his

successor (Case 4) in his new duties. An experiment had not been scheduled, but the physicist decided that the best indoctrination would be an impromptu test, even though six other people were in the laboratory. The tamper material consisted of two concentric hemispherical shells of beryllium which had been shown to bring the assembly to criticality when the bottom hemisphere measured 13 inches (O.D.) and the upper one nine inches in diameter[5] (Figure 3). In the presence of a small neutron source (10^6 neutrons/sec.), the physicist-operator placed the upper nine inch hemisphere on one inch aluminum shims above the lower hemisphere containing the plutonium sphere. Then, holding the upper hemisphere with his left thumb placed in an opening in the polar point, the operator removed the shims and allowed one edge of the upper hemisphere to be in contact with the lower hemisphere (Figure 3). Still holding the upper hemisphere, he placed a screwdriver under that part of the upper hemisphere not in contact with the lower hemisphere. He was working the screwdriver out from under the hemisphere (Figure 4) when it slipped, with the resultant criticality excursion.[5] The operator threw the shell to the floor immediately, and all personnel left the scene of the accident as rapidly as possible. One of the lesser exposed experimenters (Case 9) returned briefly to the vicinity of the assembly to drop some film badges on the assembly and to make radiation measurements. All subjects were taken to the Los Alamos Hospital for observation.

Radiation Doses

Although 85% of the energy of these chain reactions was dissipated in the form of heat,[7] the biological damage to the exposed subjects was due exclusively to three types of ionizing radiation, namely neutrons, gamma rays, and very soft x-rays and electrons (in the region of the blue glow).

Neutrons, the principal component of the incident radiation, were emitted instantaneously during the fission process. The gamma ray component was composed of prompt and delayed gamma rays. The prompt gammas were emitted during the fission process or were given off within the bodies of the subjects as a result of hydrogen capture of neutrons. The delayed gammas were emitted by the fission products and by radioactive elements induced by the incident neutrons. In these cases, the delayed gammas accounted for only a small fraction of the total dose. The radiation in the blue glow was very soft indeed. It is estimated that only 0.1% could have penetrated the tissues of the hands and arms of Cases 1 and 3. Although the intensity of these radiations in air was of the order of 7×10^7 Roentgens per second, the tissue doses were probably 3000 to 40,000 rads.[1,3]

Fortunately, it is possible to calculate the neutron doses with reasonable confidence. These doses are based on the Na^{24} activity of the serum sodium of the subjects as compared to the studies on human phantoms. At first the energy of the incident neutrons was not known, but was assumed to be 0.5 mev based on information from J. R. Oppenheimer (personal communication). By 1966 the energy spectra of the escaping neutrons had been accurately measured, thereby increasing the accuracy of the latest estimated neutron doses. Since film badges were not worn by the subjects, it is not possible to estimate the gamma doses accurately. The gamma flux with respect to distance from the assembly could be calculated knowing the number of fission reactions. The gamma ray doses were based on estimates of the time each subject spent in regions of known gamma intensity. In view of the uncertainties of the gamma ray doses, it is fortunate that they are small compared to the neutron doses. The estimated doses to the hands and arms of Cases 1 and 3 are based on studies done elsewhere of beams of

charged particles generated by accelerators. For such beams to be visualized, the radiation intensity must be of the order of 7×10^6 Roentgens per second. The estimates of hand doses (3000-40,000 rads) are based on guesses as to how long hands remained in the blue glow and on knowledge of what fraction of the radiation could penetrate the skin.

As more was learned about what happened during a critical excursion, the dose estimates were improved and in the case of neutrons became quite reliable. The evolution of the dose estimates are shown in Table 1. The first three dose calculations (in rads) were made by the late Joe Hoffman and were based on studies of Na^{24} induction by neutrons in phantoms of human bodies.. By the time of the 1967 estimates of Hankins and Hansen[8] of the Los Alamos Scientific Laboratory (LASL), the energy spectra were available for neutrons leaked by several critical assemblies and data on Na^{24} induction by neutrons as studied in human phantoms had been extended.[9] The same data were used in the 1976 and 1978 estimates of neutron dose made by James N.P. Lawrence of LASL.[10,11] Refining the neutron dose estimates resulted in an increase in dose for the five most heavily irradiated subjects, but did not change much in the rest of the subjects. Comparison of the estimated neutron and gamma ray doses shows that the radiation was predominately neutrons (except for Case 1, who received a substantial dose of delayed gammas). The composition of the incident radiation was unlike that in the Japanese irradiated by the atomic bombings of 1945. The neutron component of the radiation from the bomb in Hiroshima was only 15-30%, while that in Nagaski was almost non-existant.[12,13] When organ doses are estimated it must be remembered that the energy of the neutrons was such that they would have penetrated tissue about as well as x-rays of less than 100 kvp.[3]

The RBE for neutron exposures in the production of late effects, especially carcinogenesis, is a subject of considerable discussion and interest now. Recent studies of late effects in the irradiated Japanese indicate that the RBE for late effects in man at lower doses may be much higher than for acute radiation effects, e.g., the RBE for leukemogenesis is believed to lie between 30 and 50.([12,13]) The ICRP in Report 26[14] recommends that an RBE of ten be used for neutron exposures when the distribution of radiation collision stopping power (keV/μm) is not known at all points in the volume of interest. An RBE of 20 is recommended when the collision stopping power (keV/μm) in water is known to be 175 or above. This report on a few follow-up cases does not permit a valid statistical evaluation of these RBE values because of the small numbers involved and potential confounding factors, including hereditary influences that cannot be ruled out.

Medical Histories of the Survivors

The estimated exposure doses and main features of the medical histories of the persons irradiated in the two LASL accident are shown in Table 2. The medical histories of those persons who survived are now discussed, except for that of Case 5 who refused to cooperate with this study. The early responses (before 1950) are described in Ref. 1 and will not be repeated here.

Cases 1 and 3:

These men received the largest doses of radiation and died 24 and 9 days later respectively as a result of the exposures. The story of their acute illnesses has been fully documented in Reference 1.

Case 2:

This 29 year old military security guard was exposed to a relatively

small dose of neutrons and to a smaller dose of gamma rays (see Table 2). He returned to civilian life after the war and enjoyed excellent health for 28 years. In 1974 he suffered a heart attack from which he recovered without complications.

In March, 1976 he was hospitalized for weakness and vertigo. There were no significant physical findings, but the blood picture was distinctly abnormal. There was a pancytopenia with a leukocyte count of 2300 cells per cu mm, a hematocrit of 24%, and a platelet count of 136,000 per cu mm. Although no leukemic cells were seen in the peripheral blood smears, many myeloblastic cells were found in hyperplastic bone marrow. His disease was diagnosed as acute myeloblastic leukemia. During the next six months, the patient responded well to chemotherapy with Purinethol and repeated transfusions. Despite symptomatic improvement, the pancytopenia persisted.

By November, 1976 it had become clear that the disease was no longer being controlled by Purinethol alone. Combined chemotherapy with vincristine, prednisone, and adriamycin was begun, and this induced a satisfactory remission after some initial problems with bleeding and fever. In July, 1977 the patient's condition began to deteriorate. The leukocyte count at this time was 8400 cells per cu mm, the hemoglobin was 10 gms%, and the platelet count 33,000 per cu mm. Many immature granulocytes, including myeloblasts, were seen in the peripheral blood smear.

The patient improved somewhat after additional chemotherapy, but his clinical course was steadily downhill. His last hospital admission was in December, 1977. At that time he had a platelet count of 3000 cells per cu mm and suffered from combined renal and hepatic failure. He died January 28, 1978 at age 62, 33 years after the accident. At autopsy the diagnosis of acute myelogenous leukemia was confirmed. There was extensive leukemic

involvement of all organs and multiple hemorrhages in the heart and lung.

Case 4:

This 34 year old physicist received the highest dose of any of the survivors (see Table 2). He was partly shielded by Case 3 as a result of which his head, neck, upper torso, and right arm had a larger dose than the rest of the body.

After recovery from a six month episode of weakness following exposure, Case 4 resumed his scientific duties. Except for mild hypertension, which predated the accident, he appeared to have recovered completely from the effects of the exposure. In December, 1955 (nine years after the accident) he had a moderately severe myocardial infarction from which he appeared to recover without complications. Although his strength returned gradually, he did not feel normal and had difficulty controlling his weight. These signs and symtoms, together with an elevated blood cholesterol (488 mg) and a low PBI (1.2 mg%), led to a diagnosis of myxedema, which was treated with thyroid hormone. On this replacement therapy, the patient's condition improved and he returned to normal activity. Although his blood pressure had fallen to normal after the heart attack, his heart gradually increased in size until in 1964 roentgenographs indicated it to be 17% above the upper limits of normal. In the summer of 1966 (20 years after the accident) he suffered a fatal heart attack at age 54.

At autopsy his heart was greatly enlarged. Severe arteriosclerosis was observed in his coronary arteries as well as in the aorta. There were multiple fresh and healed infarcted areas in the myocardium. The thyroid

gland was so atrophied that it was difficult to identify. The normal thyroid tissue had been replaced by dense scar tissue in which there were multiple foci of lymphocytes and plasma cells. There were a few atrophic thyroid follicles scattered throughout the scar tissue and in the lymphoid follicles. The testes were atrophic; microscopically they showed atrophy and hyalinization of the tubular epithelium and a great increase in the interstitial fibrous tissue.

Case 6:

This 54 year old technician received a substantial dose of neutrons and gamma rays (see Table 2). Considering the magnitude of the dose, it is surprising that this man experienced no acute symptoms and was able to resume his custom of taking 20 mile week-end hikes within two weeks after the accident.

Case 6 enjoyed vigorous good health for 27 years following the accident. In 1973 and again in 1974 (when he was in his 80's) he was hospitalized for acute enteritis (diverticulitis and spastic colitis) and severe secondary anemia (2,500,000 cells per cu mm). On each occasion his condition improved after repeated transfusions.

In the summer of 1975 the patient was hospitalized for severe anemia and congestive heart failure. The anemia was first thought to be hemolytic in nature. He had a marked eosinophilia (up to 2300 cells per cu mm) and his intensely hyperactive bone marrow contained many erythroid precursors. An interesting incidental finding was a monoclonal gammapathy with marked increase in the IgG component. At this time it was thought that he might have autoimmune hemolytic anemia about to enter an aplastic crisis. His condition improved after several transfusions.

In the fall of 1975 Case 6 was again hospitalized for weakness due to severe anemia (1.7 million cells per cu mm). Physical findings were in keeping with his age, except for enlargement of his heart and liver. His leukocyte count was 17,500 cells per cu mm with a normal differential. His bone marrow was moderately hypercellular with an increase in the myeloblastoid cells showing some shift to the left. There were many immature white cells and orthochromic normoblasts. There was no increase in plasma cells. The megakaryocytes were smaller than normal and increased in number. In addition to the monoclonal gammapathy, Bence Jones proteins were now present in the urine.

Hemolytic anemia was ruled out by appropriate laboratory tests and multiple myeloma was excluded by the normal appearance of the bones on roentgenograms. It was suggested that the patient had a benign gammapathy sometimes seen in older people, but the exact nature of the refractory anemia was not known. After four transfusions the patient's condition improved and he was discharged on medication including pyridoxine, folic acid, and prednisone.

In December, 1975 the patient was readmitted to the hospital because of increasing weakness and spiking fever. His condition had deteriorated markedly and he was found to have heart failure and E. Coli septicemia. The patient had a heart attack while in the hospital and died just before Christmas, 1975 at age 83 (29 years after the accident).

At autopsy, a vegetative bacterial endocarditis was found as well as E. Coli septicemia and cardiac and splenic infarcts secondary to emboli. Microscopically, the bone marrow was moderately hyperplastic with a marked increase in red cell precursors and some increase in plasma cells. The autopsy gave no clues as to the nature of the anemia.

Case 7:

This 21 year old soldier was exposed to an intermediate dose of mixed radiation (see Table 2). When last examined 28 months after the accident, there were no significant physical or laboratory findings. This man was killed in combat in Korea in 1952 at age 27.

Case 8:

This 23 year old college graduate received a relatively small dose of mixed radiation (see Table 2). After the war he returned to graduate school and became an important executive in industry.

Case 8 enjoyed excellent health until 1964 (18 years after the accident). At this time he developed what was first thought to be acute sinusitis. When antibiotics failed to relieve his symptoms, hemotalogical studies revealed a leukocyte count of 300,000 cells per cu mm with many blast cells seen in the blood smear. His bone marrow was hypercellular and contained many immature myeloid cells. A diagnosis of acute myelogenous leukemia was made and he was started on 6 mercaptopurine (6 MP) therapy. He had a satisfactory remission for five months. At this time his medication was changed to methotrexate. At first he seemed to respond, but soon his condition worsened. In March, 1965 his lymph nodes and spleen were palpable, and he developed a superficial ulcer on his palate. His leukocyte count was 22,700 cu per mm; his hematocrit was 21.5%, and his platelet count was 16,000 per cu mm. Many immature granulocytes and an occasional myeloblast were seen in the blood smears. Erythroid elements and megakarocytes were rarely seen in the bone marrow.

After an unsatisfactory trial with prednisone and vincristine, the patient entered the hospital of the Brookhaven National Laboratory in April, 1965 for treatment with extracorporeal irradiation (e.c.i.) of

his blood. Shortly after admission he developed a severe headache with neurological signs which were completely relieved by intrathecal methotrexate. Following daily e.c.i. treatments, the leukocyte count (then 79,000) fell rapidly and stabilized at 30,000 cells per cu mm. His spleen decreased in size and the patient felt better. In May, 1965 subcutaneous nodules developed and his spleen and liver became larger. He was given two series of treatments with tritiated thymidine in addition to the e.c.i. His leukocyte count fell to 800 cells per cu mm and the lymphoid masses shrank in size. His clinical condition went downhill rapidly and he died after a grand mal seizure on July 18, 1965. Death occurred at age 42, 19 years after the accident.

At autopsy, extensive leukemic infiltration resembling a sarcomatous growth of all organs, soft tissues, and meninges was observed.

Case 9 amd 10:

These young men, 36 and 23 years old respectively, received small doses of mixed radiation (see Table 2). When last examined in 1978 (32 years after the accident), they were found to be in excellent health and spirits. They showed no physical or laboratory evidence of late radiation injury.

Discussion:

Since ionizing radiations, particularly neutrons are known to be leukemogenic, it is tempting to attribute the terminal illnesses of Cases 2 and 8 to their accidental radiation exposure. Similarly, since the thyroid gland is readily destroyed by radiation,[15] one might suspect that the myxedema of Case 4, presumably radiation induced, may have promoted his coronary disease by elevating the blood cholesterol. This could well have precipitated the first heart attack. By the same line of reasoning, one

can imagine that the insult to the hematapoietic tissue of Case 6 resulting from the radiation exposure could have been a factor in the development of the terminal refractory anemia.

Our statistician friends, however, assure us that it is dangerous to draw definite conclusions from such small numbers of cases in such limited populations, as there may be etiologic factors other than radiation exposure. For example, a brother of Case 2 also died of leukemia (and three other siblings are believed to have had cancer). This makes it likely there is a familial component to the development of the disease. Similarly, the father of Case 4 also had a myocardial infarction when he was in his early 40's. This also suggests a genetic factor. And finally, in the case of the refractory anemia of Case 6, this disease (in the absence of leukemia) is not usually considered to be a late effect of radiation exposure. Furthermore, if one reviews the acute hematological and bone marrow reactions of Case 4, there is not much evidence for a severe insult to the hematopoistic system except for a mild persistant lymphopenia.

In conclusion, it should be pointed out that these accidental total body exposures (non-homogeneous in the case of the most heavily exposed subjects) were unique in that moderately fast neutrons accounted for almost all of the exposure doses. Of the six survivors followed for more than ten years after the accident, four died 19 or more years after the accident. It seems likely that the two leukemic deaths represent late effects of the radiation exposure although one may have had a genetic component predisposing to leukemia. The other two deaths could conceivably have been related to the exposures, although one also seems to have a genetic factor. The two survivors were in good health in 1978 and showed no clinical or laboratory evidence of late radiation damage.

FIGURE CAPTIONS

FIGURE 1: Mock-up of Accident LA-1: Assembly before accident.

FIGURE 2: Mock-up of Accident LA-1: Assembly after accident.

FIGURE 3: Mock-up of Accident LA-2: Assembly before accident.

FIGURE 4: Mock-up of Accident LA-2: Assembly just before accident.

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TABLE 1

Evolution of Total Body Radiation Dose

Rads of Neutrons and Gamma Rays

Year of Estimate	1948	1952	1957	1968	1976	1978
Reference	6	1	3	8	10	11
Case 1						
N*	96	96	168	-	200	200
	110	110	487	-	200	110
Case 2						
N	6	6	9	-	7-8	8
	0.1	0.1	3	-	2	0.1
Case 3						
N	386	386	407	1000	-	1000
	114	114	156	100	-	114
Case 4						
N	78	78	82	166	-	166
	26	26	41	17	-	26
Case 6						
N	37	37	42	51	-	51
	11	11	19	5	-	11
Case 7						
N	28	28	20	20	-	33
	9	9	10	2	-	9

(Continued)

TABLE 1 (Continued)

Case 8						
N	11	11	11	8-20	-	12
	4	4	6	1-2	-	4
Case 9						
N	8	8	9	9	-	9
	3	3	4	1	-	3
Case 10						
N	7	6	6	7	-	7
	2	2	3	1	-	2

* N = neutrons

** = gamma rays

TABLE 2

SUMMARY OF CLINICAL COURSE OF SUBJECTS

CASE	Age at Exposure (yrs.)	Estimated (1978) In Rads Neutrons	Dose Rads Gamma	Acute Radiation Response	Age (yrs.) when last seen or age at death (d)	DIAGNOSIS
L.A. Accident #1						
1	26	200	110	Severe* leading to death in 24 days	26 (d)	Fatal Acute Radiation Syndrome (hematopoetic type)
2	29	8	0.1	None (death in 32 years)	62 (d)	Fatal Acute Myeloblastic Leukemia
L.A. Accident #2						
3	32	1000	114	Severe* (death in 9 days)	32 (d)	Fatal Acute Radiation Syndrome (Gastrointestinal type)
4	34	166	26	Moderate, severe fatigue for 6 mos: epilation, aspermia (death in 20 yrs.)	54 (d)	Fatal Myocardial Infarction (Myxedema, compensated,cataracts)

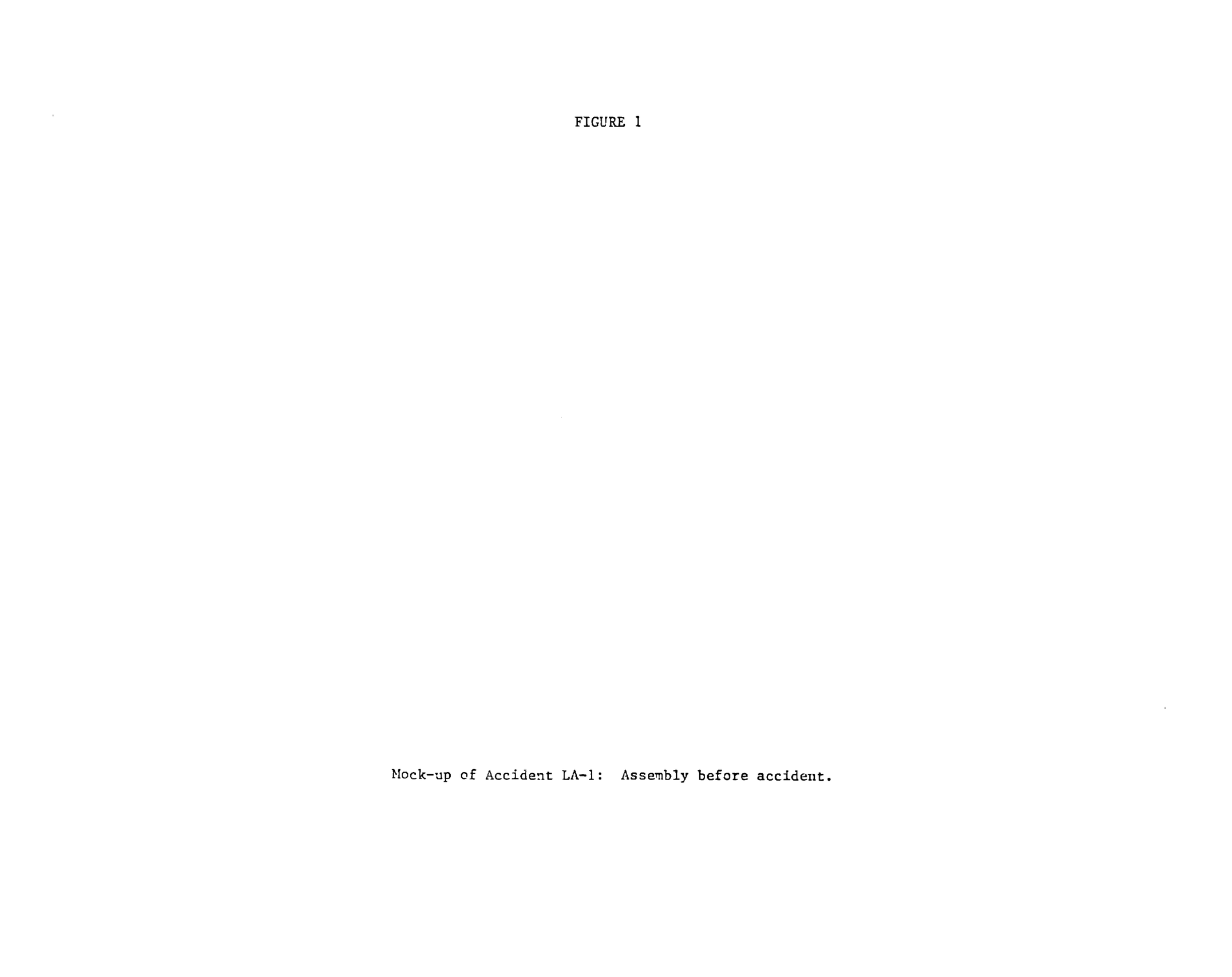
(Continued)

TABLE 2 (Continued)

5	Refused consent to follow-up					Alive in 1978
6	54	51	11	None	83 (d)	Clinical Aplastic Anemia, Fatal Bacterial Endocarditis
7	21	33	9	None	27 (d)	Korean · Fatality
8	23	12	4	None (death in 18 years)	42 (d)	Fatal Acute Myelocytic Leukemia
9	36	9	3	Transient Nausea	57	Healthy Male
10	23	7	2	None	55	Healthy Male

* See reference 1

FIGURE 1



Mock-up of Accident LA-1: Assembly before accident.

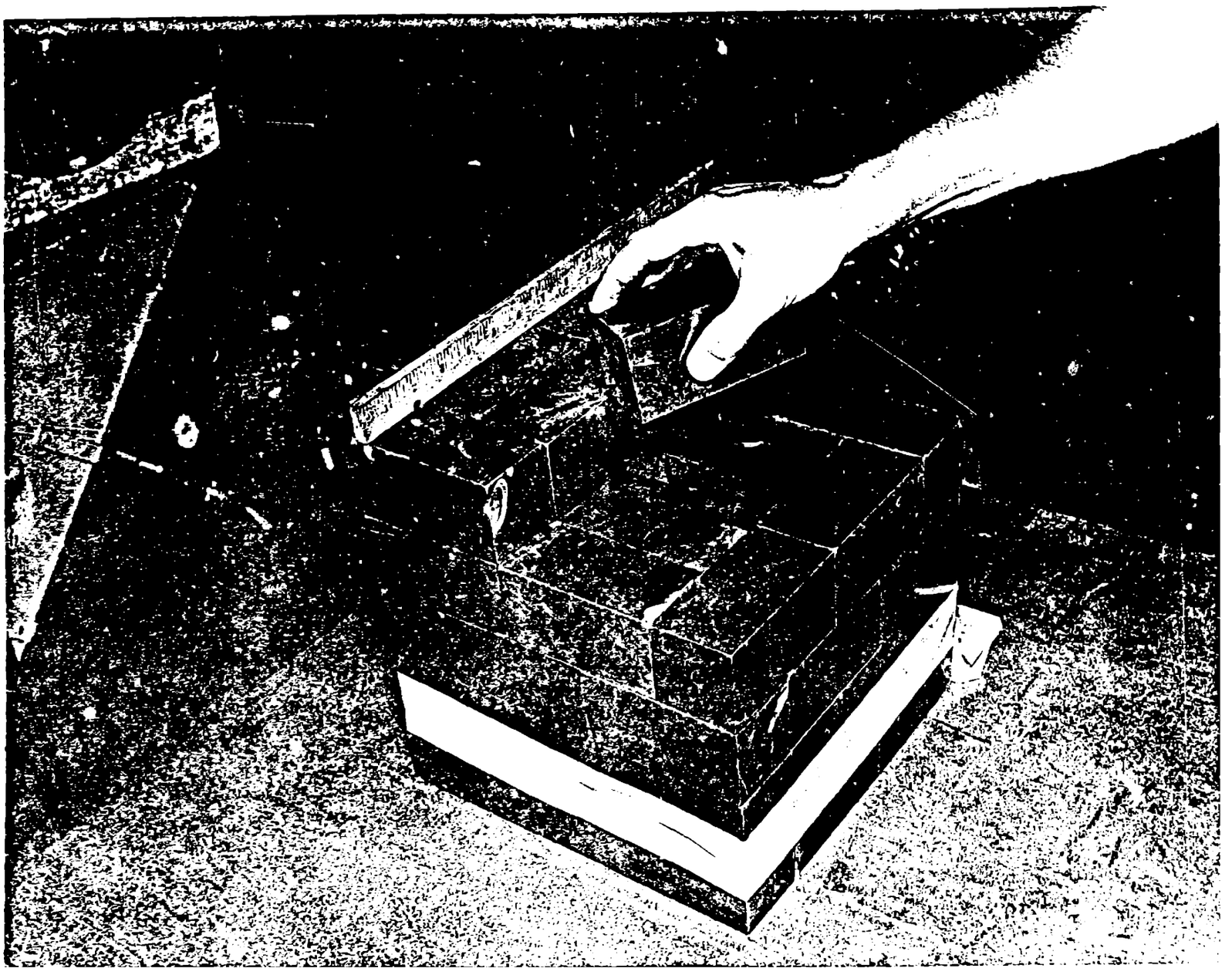


FIGURE 2

Mock-up of Accident LA-1: Assembly after accident.

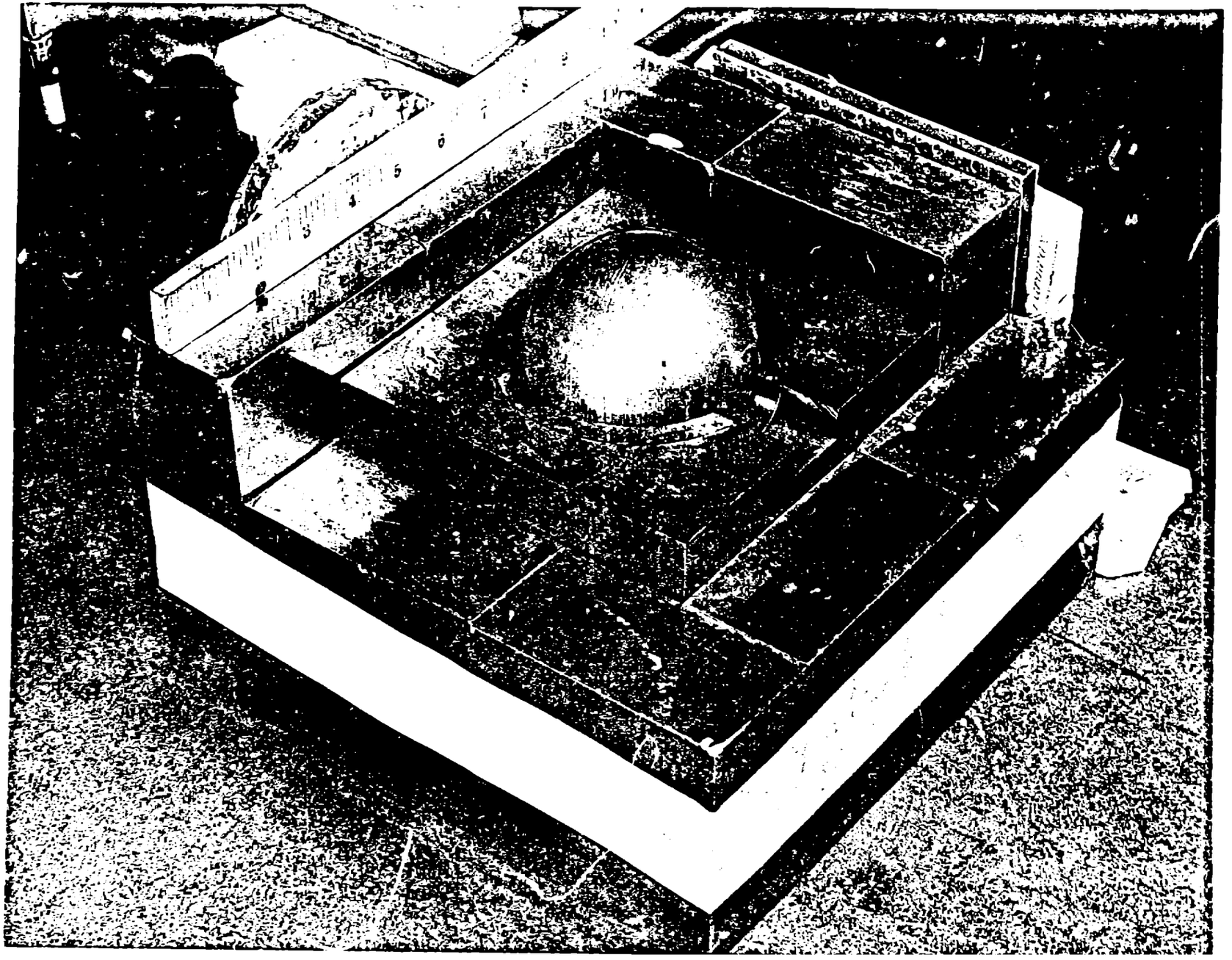


FIGURE 3

Mock-up of Accident LA-2: Assembly before accident.

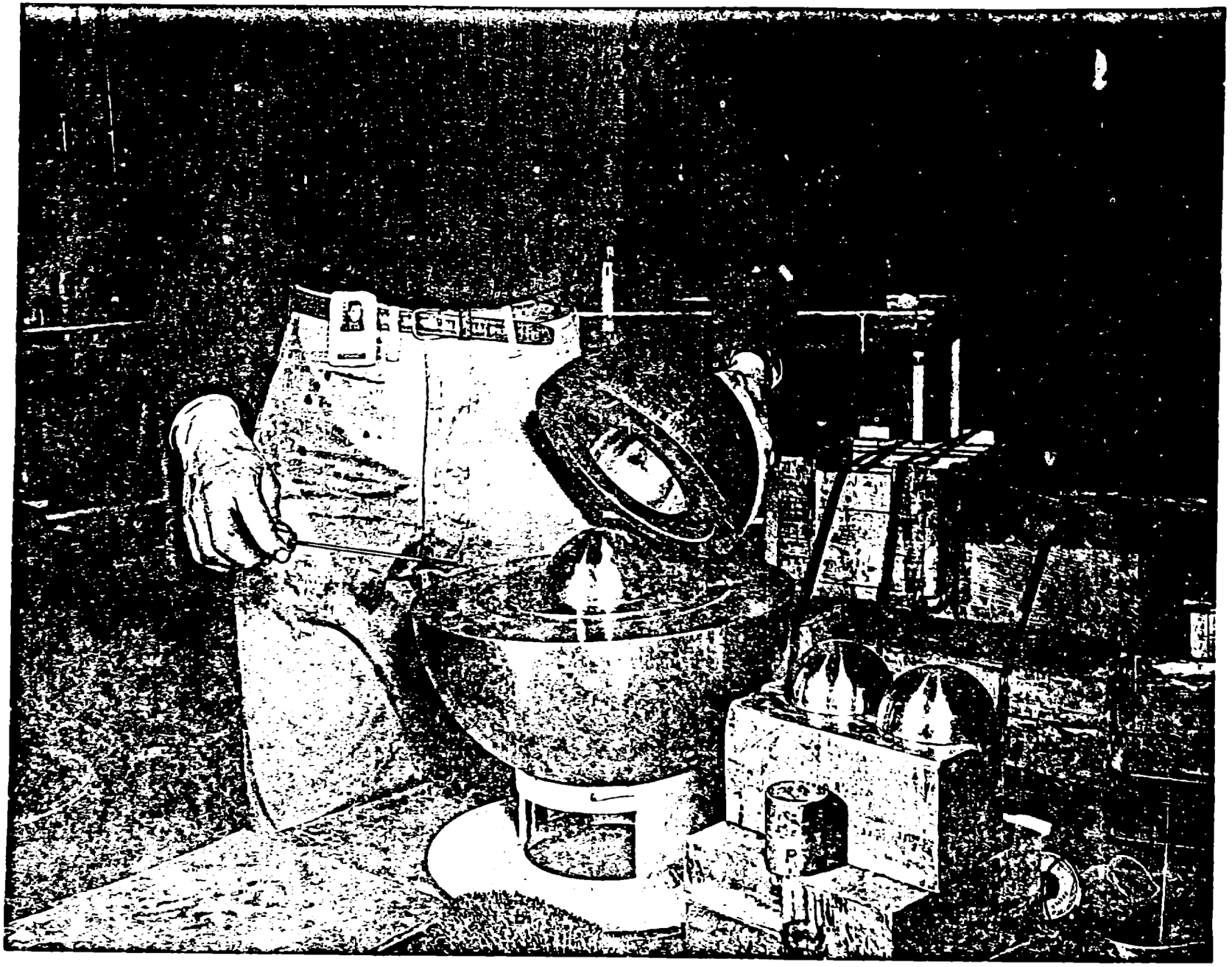
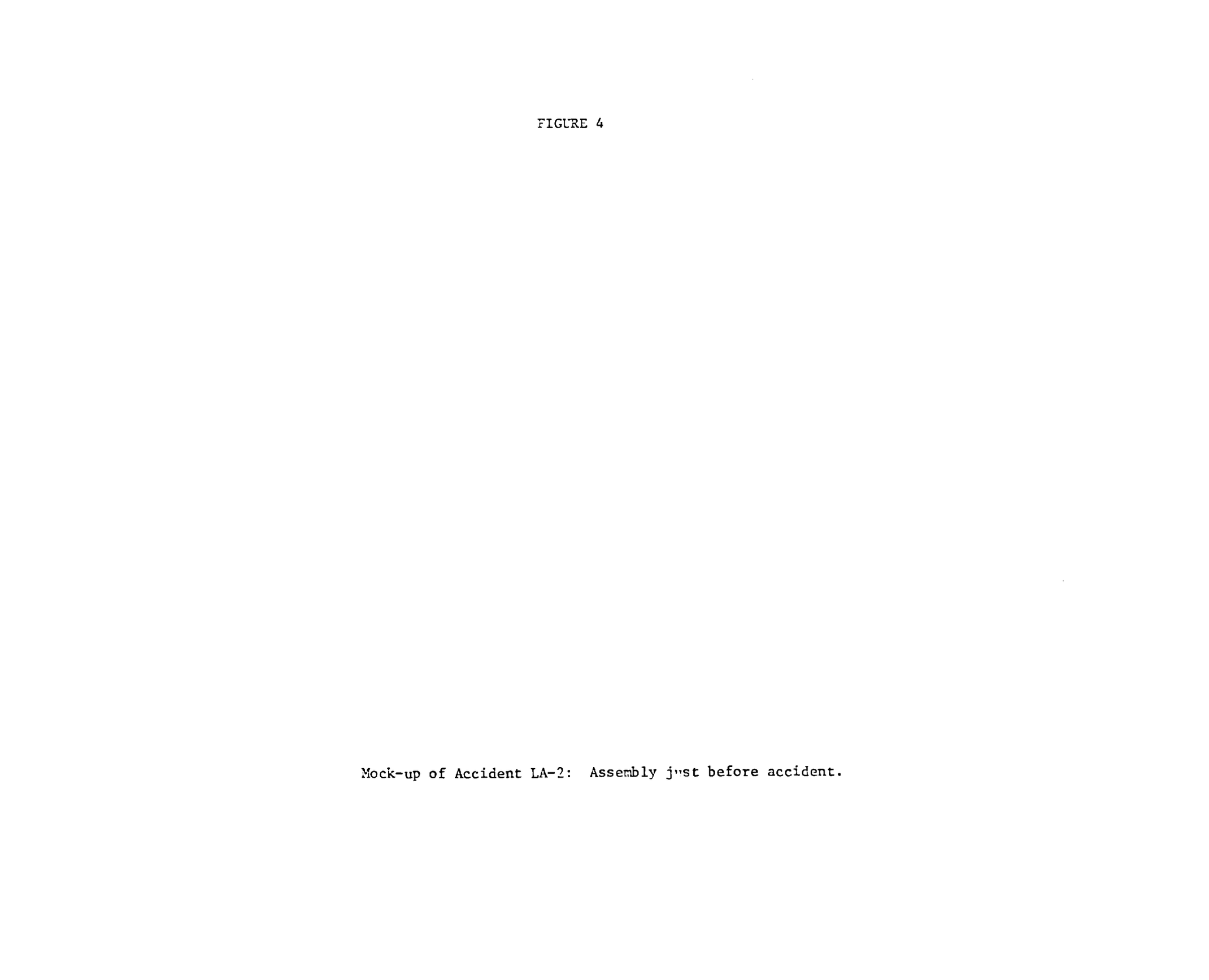


FIGURE 4



Mock-up of Accident LA-2: Assembly just before accident.